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A Brief Note on Antiepileptic Drugs

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Commentary

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ABOUT THE STUDY

Anticonvulsant drugs, often known as Antiepileptic Drugs (AEDs), are commonly divided into "generations" based on when they were discovered and introduced. The pharmacologic properties and clinical use of second- and third-generation AEDs is the subject here. Since 1990, three-quarters of the medications we use on a daily basis have been developed and brought to market: they are known as second and third-generation AEDs. Beginning in 1989, second-generation "designer" AEDs was developed during a 15-year span, while third-generation AEDs were introduced in 2008.

Clobazam, eslicarbazepine acetate, ezogabine/retigabine, felbamate, gabapentin, lacosamide, lamotrigine, levetiracetam, oxcarbazepine, perampanel, pregabalin, rufinamide, topiramate, vigabatrin, and zonisamide are a few examples. Anticonvulsants differ from other medications in that they are not categorised by mechanism

of action, but rather simply lumped under the title of anticonvulsant due to the fact that each drug's method of action is both unknown and possibly numerous in nature.

Valproic acid has been shown to have anticonvulsant properties in a variety of epilepsy models. Valproate increases GABA levels in synaptosomes, possibly by preventing its breakdown; it boosts the postsynaptic response to GABA; and it increases potassium conductance, causing neuronal hyperpolarization, at lower concentrations. Valproate inhibits the firing of 5-HT neurons in the dorsal raphe, which are involved in pain modulation.

Migraine prevention is achieved with the use of divalproex sodium and sodium valproate. Each of these drugs was found to be significantly more effective than placebo in lowering headache frequency in two placebo-controlled trials. Extended-release divalproex sodium 500-1000 mg (once a day) significantly reduced the mean 4-week migraine headache rate compared to placebo in a double-blind, randomised, controlled trial. Valproic acid is a simple 8-carbon, 2-chain fatty acid having an oral bioavailability of 80 percent. It's strongly protein-bound and has an elimination half-life of 8 to 17 hours.

The most common side effects of valproate include nausea, vomiting, and gastrointestinal distress. These are usually self-limiting and occur less frequently with divalproex sodium than sodium valproate. The incidence of gastrointestinal complaints reduces when the medication is prolonged, especially after 6 months. Tremor and baldness may develop later. Valproate has limited influence on cognitive function and induces drowsiness only in rare cases. Valproate administration has been linked to severe side effects such hepatitis and pancreatitis on rare occasions. The amount of concurrent medications used, the patient's age, the presence of genetic and metabolic abnormalities, and the patient's overall health all influence the frequency. These peculiar reactions are unpredictably unpredictable.

Valproate is teratogenic and should not be used by pregnant or planning to become pregnant women. Hyperandrogenism, which is caused by high testosterone levels, ovarian cysts, and obesity, is a major issue in young epileptic women who take valproate. Pregnancy and a history of pancreatitis or a hepatic condition, such as chronic hepatitis or liver cirrhosis, are absolute contraindications to valproate. Hematological problems, such as thrombocytopenia, pancytopenia, and bleeding disorders, are also important contraindications.

Valproic acid is available in 250 mg capsules and 250 mg/5 ml syrup form. Divalproex sodium is a stable coordination complex made up of a 1:1 molar ratio of sodium valproate and valproic acid. Divalproex sodium is available as 125, 250, and 500-mg capsules, as well as a sprinkle formulation, in an enteric-coated form. Begin with 250-500 mg each day, divided into two doses, and gradually raise the amount. A maximum dose of 60 mg/kg/day is suggested. The efficacy of an extended-release version of divalproex sodium was comparable to that of the tablet formulation. The clinical trial's adverse event profile, on the other hand, revealed nearly comparable rates of adverse events for the placebo and active treatment groups.